REMARKS

Upon entry of this amendment, claims 19, 20 and 25-38 are pending in the instant application. Claims 1-18 and 21-24 have been cancelled without prejudice or disclaimer. Claims 19, 20 and 25 have been amended, and claims 26-31 have been added. The present amendments and new claims are fully supported by the specification and the claims as originally filed. For example, support for the amendments to claims 19, 20 and 25 and new claim 26 is found at least at page 10, lines 24-30; and in Example 7 at pages 17-18. Support for new claims 27 and 28 is found at least at page 3, lines 11-18, while support for new claims 29 and 30 is found at least in Example 1 at pages 11-12. Support for new claim 31 is found at least at page 4, lines 3-5. Support for new claims 32-38 is found at least in Example 2 at page 14, lines 5-21. Accordingly, no new matter has been added by the amendments presented herein.

Information Disclosure Statement

The Examiner has indicated that the listing of references in the specification is not a proper information disclosure statement. Applicants submit herein a Supplemental Information Disclosure Statement, along with the appropriate fee in accordance with the provisions of 37 C.F.R. §§ 1.97(c), 1.98. Applicants, therefore, request that the Examiner consider the references cited on the enclosed modified PTO-Form 1449.

Specification

The Examiner has objected to the Title of the Invention as "not descriptive". The Title of Invention, on page 1, lines 4-5, has been amended herein to read "Methods for Diagnosing and Treatment of Hyperphosphatemic Conditions Using FGF-20 Polypeptides". Applicants submit that the amended title is descriptive and reflects the subject matter recited by the pending claims. As such, this objection should be withdrawn.

The Examiner has also objected to informalities in the specification at pages 17-18. According to the Examiner, Example 7 does not comply with 37 C.F.R. § 1.821(d), which requires the use of a sequence identifier when making reference to a sequence. The specification at pages 17-18 has been amended herein to include a sequence identifier, *i.e.*, SEQ ID NO:1, for the amino acid sequence presented in Example 7. Accordingly, withdrawal of this objection is requested.

With regard to page 3 of the originally filed specification, Applicants note that the file copy of the instant application contains a complete page 3. A copy of page 3 from Applicants' files is included herewith for the Examiner's convenience.

Claim Rejections Under 35 U.S.C. § 112, First Paragraph

Claim 20 has been rejected under 35 U.S.C. § 112, first paragraph for lack of written description. According to the Examiner, "there is no disclosure in the instant specification regarding instability of FGF20 or ways to modify FGF20 which would increase stability." (Office Action, page 4).

Dependent claim 20 has been amended herein to recite an isolated polypeptide that consists of the amino acid sequence of SEQ ID NO:1. Moreover, the amended claims presented herein do not recite FGF20 polypeptides that comprise a mutation that confers increased stability to the FGF20 polypeptide. Accordingly, Applicants submit that this rejection no longer applies to the pending claims and, therefore, should be withdrawn.

Claim Rejections Under 35 U.S.C. § 112, Second Paragraph

Claims 19-20 and 25 have been rejected under 35 U.S.C. § 112, second paragraph as being indefinite. According to the Examiner, "the mere recitation of the term 'FGF20' does not convey the actual nature of the molecule being administered." (Office Action, page 3).

The pending claims have been amended herein to recite the structure of the molecule being administered. In particular, claim 19 has been amended to recite a method of treating a hyperphosphatemic condition in a mammal, the method comprising administering to a mammal afflicted with the disorder a therapeutically effective amount of an isolated polypeptide comprising the amino acid sequence of SEQ ID NO:1. Claim 25, as amended, is directed to a method of treating a condition involving deposition of calcium and phosphate in the arteries or soft tissues of a mammal, the method comprising administering to the mammal a therapeutically effective amount of an isolated polypeptide comprising the amino acid sequence of SEQ ID NO:1.

Applicants, therefore, submit that the amended claims are clear, definite and complete. As such, withdrawal of this rejection is requested.

CONCLUSION

Applicants respectfully submit that the pending claims are in condition for allowance. If there are any questions regarding these amendments and remarks, the Examiner is encouraged to contact the undersigned at the telephone number provided below.

Respectfully submitted,

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growth in children may also be due to phosphate depletion. Hypophosphatemia also results in decreased levels of 2,3-diphosphoglyceric acid and adenosine triphosphate in red blood cells that in turn alter the dissociation of oxyhemoglobin so that less oxygen is delivered in the periphery which mechanism my explain the central nervous system dysfunction seen in hypophosphatemia patients..

Negative phosphorus balance is rarely caused by inadequate phosphorus adsorption I n the intestine. Maintenance of normal phosphorus balance is dependent upon efficiency of renal excretion of conservation. In severe renal failure, hyperphosphatemia results from inadequate renal phosphorus clearance; heritable or acquired renal tubular defects may lead to hypophosphatemia due to inadequate renal conservation of phosphorus.

Hyperphosphatemias defined in adults as an elevation of serum phosphorus above 5 mg/dL. Unfortunately, the condition produces no direct symptoms. However, with maintenance of high phosphorus levels for long periods of time, the driving force for mineralization is increased, and calcium phosphate may be deposited in abnormal sites. Severe hyperphosphatemia is normally associated with extensive cellular or tissue damage. The combination of an increased release of phosphate from damaged muscle tissue and an impaired ability to excrete phosphorous secondary to renal failure (the most common cause of hyperphosphatemia) causes moderate to severe hyperphosphatemia.

Hyperphosphatemia with levels of to 40 mg/dL and above, has occurred secondary to increase absorption from the intestines following administration of excess phosphate salts orally or from the colon as a result of enemas containing phosphate salts. Overmedication with vitamin D, and its production by granulomatous tissue in diseases such as sarcoidosis and tuberculosis may cause hyperphosphatemia. Lactic acidosis is especially important as a cause of hyperphosphatemia. Other causes of hyperphosphatemia may be found in pathologies involving decreased renal excretion such as in the case of renal insufficiency, hypoparathyroidism, hyperostosis, adrenal insufficiency, and infantile hypooshosphatasis; involving intestinal absorption such as in vitamin D ingestion, and granulomatous diseases producing vitamin D (as, for example, tuberculosis); involving internal redistribution of phosphorus such as lactic acidosis, reduced insulin level, acute respiratory acidosis, and lactic acid infusion; involving cellular release of phosphorus such as rhabdomyolysis, tumor lysis, and acute hemolysis;